Finger pad tophi in a patient with Raynaud phenomenon

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INTRODUCTION

The natural history of gout includes 4 progressive stages (asymptomatic hyperuricemia, acute arthritic attacks, intercritical gout, and chronic gouty arthritis).[1] Asymptomatic hyperuricemia is characterized by elevated serum urate. Deposition of monosodium urate (MSU) crystals in articular and periarticular tissues then instigates acute arthritic attacks. Intercritical gout eventually results in chronic gouty arthritis with no pain-free intervals.

Tophi, MSU crystal aggregates, are primarily associated with chronic gout. Common locations for tophus development include the helix of the ear, olecranon bursa, hands, knees, feet, and fingers.[2,3] There are few reports of tophaceous deposits on the fingertips. Finger pad tophi have been reported in individuals without prior acute gouty arthritis. Finger pad tophi represent a dermatologic presenting feature of gout and an indication for prompt initiation of urate-lowering therapy (ULT) to prevent sequelae of chronic hyperuricemia.[3]

We describe a case of finger pad tophi in the context of chronic Raynaud phenomenon and suggest a potential role for the Raynaud phenomenon in the process of tophus formation.

Abbreviations used:

MSU: monosodium urate
ULT: urate-lowering therapy

REPORT OF CASE

A 93-year-old woman with a 6-month history of yellow-white fingertip lesions was referred to our clinic to rule out calcinosis cutis. She had a history of cerebrovascular accident, chronic renal failure, atrial fibrillation, and hypothyroidism. She acknowledged a history of chronic Raynaud phenomenon described as complete and painful whitening of her fingers that occurred mostly in the winter. She denied symptoms of inflammatory arthritis.

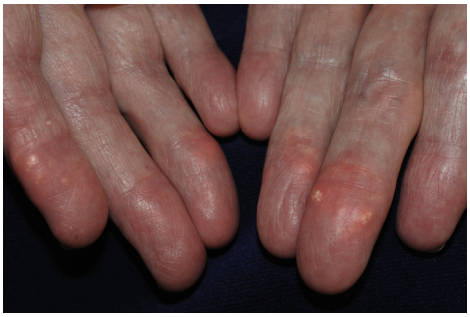

Fig 1. Tophaceous gout. Grouped white-to-yellow papules on the finger pads.

Medication use included levothyroxine, 25 μg daily, furosemide, 20 mg daily, atenolol, 25 mg daily, hydrochlorothiazide, 37.5 mg daily, and spironolactone, 37.5 mg daily. She had been started on colchicine, 0.6 mg twice daily, 1 month before our assessment for presumptive treatment of either calcinosis cutis or gout.

Blood pressure was 110/80 mm Hg. Dermatologic examination found grouped and distributed white-to-yellow milialike papules measuring 2 to 3mm in diameter on the distal finger pads of the index and long fingers, which were tender to palpation (Fig 1). Examination of the proximal nail folds found a normal capillary pattern. There was no cutaneous sclerosis or tapering of the digits.

Radiograph of the hands showed periarticular calcifications at both first carpometacarpal joints and

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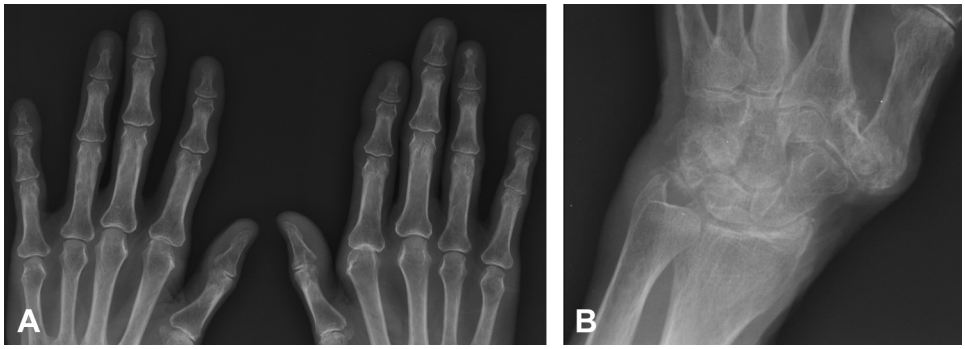

Fig 2. Radiograph of the hands. **A,** Soft tissue swelling along with periarticular calcifications at both 1st carpometacarpal joints. There is no digital calcification. **B,** Calcification in the region of the triangular cartilage on the left and adjacent to the triquetrum and ulnar styloid process of the left hand.

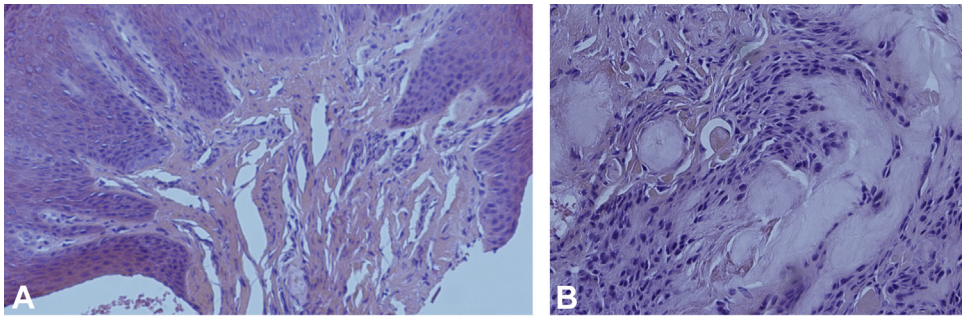

Fig 3. Tophaceous gout. Amorphous eosinophilic deposits in dermis show characteristic clefting. Original magnifications: **A,** ×10 and **B,** ×20.

adjacent to the ulnar styloid process, evidence of distal osteoarthritis, and soft tissue swelling of the finger pads without digital calcification (Fig 2). Serum creatinine level was 147 μmol/L, estimated glomerular filtration rate was 26 μmol/L, blood urea nitrogen level was 15.6 mmol/L, and serum urate concentration was 695 μmol/L (normal, 140−360). Calcium (2.52 mmol/L) and phosphate (0.9 mmol/L) levels were normal. The presentation was consistent with finger pad tophaceous gout. Diagnosis was confirmed by a skin biopsy (Fig 3).

Amlodipine, 2.5 mg daily, was started for treatment of Raynaud phenomenon. ULT using allopurinol was initiated.

DISCUSSION

The differential diagnosis of white papules or nodules on the finger pads includes gout, calcinosis cutis, chondrocalcinosis (pseudogout), pyogenic pustules, and oxalosis. Calcinosis cutis involving the digits is noted in patients with systemic sclerosis. These patients have evidence of capillary dropout along the proximal nail folds, sclerosis or tapering of the digits, and pitted scars on the fingertips.[4] Patients with pseudogout or oxalosis may rarely present with tumoral calcifications of the digits but not with punctuate white deposits. Any of these conditions would result in radiographic calcifications.

Diagnosis is confirmed by biopsy and histopathologic examination. Gout tophi manifest as a dermal or subcutaneous granulomatous reaction with macrophages and foreign body giant cells.[5] Samples must be preserved in alcohol to visualize brown needle-shaped crystals, as crystals will dissolve in formalin leaving amorphous eosinophilic deposits with characteristic clefts (Fig 3).[5] Alternatively, needle aspirate of tophi can be examined with polarizing microscopy[3]; gout crystals will demonstrate negative birefringence. Dual-energy computed tomography represents a newer diagnostic tool with excellent sensitivity for detection of MSU crystal deposits in tophaceous gout[6]: Compositions of tissues are determined by analyzing the difference in attenuation of materials simultaneously exposed to 2 different x-ray spectra, allowing the direct identification and visualization of MSU crystals. Pertinent investigations include radiography (peri- or intra-articular soft tissue masses and/or erosions[7]), serum uric acid (hyperuricemia), blood urea nitrogen, serum creatinine, and estimated glomerular filtration rate (renal dysfunction[2]).